ONION TOXICOSIS IN BUFFALOES

N.A. Patil*, Ajay Satbige, Yallappa S. Ingale, Sandeep H. and Vivek R. Kasaralikar

ABSTRACT

Nine buffaloes non-descript aged between 4 to 9 years affected by onion toxicosis were presented to Veterinary College, Hospital, Bidar, with the signs of anorexia, dullness, depression, staggering gait and passing dark brown coloured urine. On clinical examination affected buffaloes revealed tachycardia, polypnoea, pale mucous membrane, and coffee decoction color urine. Hematological examination revealed decreased values of total erythrocyte count, hemoglobin concentration and packed cell volume with leukocytosis. Erythrocyte indices revealed tendency towards macrocytic hypochromic anemia. Serum biochemistry revealed normal serum urea nitrogen and total serum bilirubin. Affected buffaloes were treated with vitamin E and Selenium along with supportive therapy of 5% dextrose and 8 buffaloes were recovered within 5 days of treatment. Necropsy findings of dead buffalo revealed pallor of tissue, jaundice, dark brown kidneys and urine and fragments of onion in rumen.

Keywords: *Bubalus bubalis*, buffaloes, onion toxicosis, non-descript buffaloes, haemoglobinuria, Vitamin E, Selenium

INTRODUCTION

Hemolytic anemia caused by spontaneous ingestion of onion (*Allium cepa*) has been reported in buffaloes (Borelli *et al*., 2009) and in cattle (Hutchison, 1977; Hothi *et al*., 1980; Chakrabarti and Basak, 1994; Rae, 1999; Van der Kolk, 2000; Bhikane *et al*., 2011). Onion toxicosis occurs when ruminants are grazed mainly on cultivated onions. The present paper reports occurrence of onion toxicosis in buffaloes, clinicopathological features and its management with antioxidants.

CASE HISTORY

Nine buffaloes non-descript aged between 4 to 9 years were presented to Veterinary College Hospital, Bidar with the complaint of passage of dark brown coloured urine. The history revealed that animals were grazed on cultivated onions (*Allium cepa*) for last 3 to 5 days. Affected buffaloes were found anorectic, dull, depressed with staggering gait.

RESULT AND DISCUSSION

On clinical examination tachycardia (85 to 90/minutes), polypnoea (34 to 38/ minutes),

Department of Veterinary Medicine, Veterinary College, Bidar, Karnataka, India, *E-mail: napvety@gmail.com

pale mucus membrane, passing constipated faeces and dark brown coloured urine were observed. The blood smear examination revealed absence of any hemoprotozoan infection. Hematological examination revealed decreased total erythrocyte count (3.42±0.24 million/ cumm), decreased hemoglobin concentration (4.77±0.29g/dl), decreased packed cell volume (19.44±0.96%) and slightly elevated leukocyte count (10.21±5.61x10^3µl). Erythrocyte indices revealed slightly elevated MCV (59.59±5.58 fl) and decreased MCHC (14.60±1.53g/dl) indicated tendency towards macrocytic hypochromic anemia. Serum biochemistry revealed serum urea nitrogen (8.21±0.58 mg/dl) and total serum bilirubin (0.46±0.08 mg/dl) values within normal range.

Affected buffaloes were treated with Vitamin E and Selenium injection (Rapranol, Cadilla) 1 ml/25 Kg B.wt. I/M on alternate day for 4 days along with supportive therapy of 5% dextrose. Eight buffaloes were recovered clinically within 5 days of treatment. One buffalo died on 3rd day and necropsy findings of pallor of tissues, jaundice, dark brown kidneys and urine and fragments of onion in rumen were observed.

Bovines are more susceptible to onion toxicity when compared to other species. The toxic principle of domestic onion is n-propyl disulphide, an alkaloid. The oxidative threat is greater in ruminants because onion also contain S-methyl cysteine Sulfoxide (SMCO). The SMCO are hydrolysed in the rumen by anaerobic bacteria to thiosulfonate which is then further metabolized to dipropyl disulfides and dipropenyl disulfides. These disulfides are responsible for oxidative damage in erythrocytes which results in precipitation and aggregation of hemoglobin and its binding to the cytoplasmic membrane forming Heinz bodies. The formation of Heinz bodies increases erythrocyte fragility and extra vascular haemolysis. Damaged erythrocytes are removed from circulation leading to hemolytic anemia, haemoglobinuria and haemoglobinemia.

Borelli *et.al* (2009) and Bhikane *et al.* (2011) reported similar clinical signs in buffaloes and bullocks with onion toxicosis. Salgado *et al.* (2011) indicated the use of antioxidant vitamins to treat onion poisoning in animals. Vitamin E and Selenium containing enzyme Glutathione peroxidase has long been recognized as a natural biological antioxidant and are integral part of the cellular antioxidant system in most mammalian cells (Radostits *et al*., 2007). Bhikane *et al.* (2011) reported successful treatment of onion poisoning in bullocks with Vitamin E and Selenium which is in agreement with the results of the present study. Hence it is concluded that Vitamin E and Selenium may be effectively used to treat onion poisoning in buffaloes.

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